

Chyloperitoneum After Laparoscopic Roux-en-Y Gastric Bypass (LRYGB)

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Abstract A true chylous effusion is defined as the presence of ascitic fluid with high fat (triglyceride) content, usually higher than 110 mg/dl. We report a case of chyloperitoneum following laparoscopic Roux-en-Y gastric bypass (LRYGB) in a 40-year-old patient who was admitted for surgery on May 31, 2007. On August 2008 an abdominal CT with contrast was ordered for chronic abdominal pain showing diffuse ascites as well as mesenteric adenitis. On September 2008, the patient was admitted to the hospital. An elective diagnostic laparoscopy was scheduled. A large amount of chylous fluid was found. Microscopic analysis came back negative. The patient made an uneventful recovery after surgery. To our knowledge, this is the first reported case of chylous ascites following LRYGB. Chyloperitoneum should be considered as a possible cause of ascites in patients with chronic small bowel obstruction following a LRYGB.

Keywords Chyloperitoneum · Obesity · Bariatric surgery · Laparoscopic Roux-en-Y gastric bypass

Chylous ascites can present de novo or as a secondary event. We present a case of chylous ascites following a laparoscopic Roux-en-Y gastric bypass (LRYGB). After imaging studies, a diagnostic laparoscopy was performed; the patient was successfully treated for chyloperitoneum with no need to convert it into an open procedure.

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Introduction

The extravasation of milky chyle into the peritoneal cavity is known as chylous ascites. A true chylous effusion is defined as the presence of ascitic fluid with high fat (triglyceride) content, usually higher than 110 mg/dl [1]. This can occur de novo as a result of trauma, neoplasm, obstruction of the lymphatic system, as well as many other causes that have been described in the literature.

The most common etiologies in Western countries are abdominal malignancies and cirrhosis. In Eastern and developing countries, infectious etiologies, such as tuberculosis and filariasis, account for most cases. Three mechanisms have been postulated to cause chylous ascites: (1) exudation of chyle from dilated lymphatics on the wall of the bowel and in the mesentery caused by obstruction of lymphatic vessels at the base of the mesentery or the cisterna chili (e.g., by malignancies); (2) direct leakage of chyle through a lymphoperitoneal fistula (e.g., those which develop as a result of trauma or surgery); and (3) exudation of chyle through the wall of dilated retroperitoneal lymphatic vessels (e.g., in congenital lymphangiectasia or thoracic duct obstruction) [2]. Delayed presentations following surgery can occur if the mechanism of ascites formation is adhesion-induced lymphatic obstruction rather than lymphatic vessel disruption [3].

Case Presentation

We report a case of chyloperitoneum following a laparoscopic Roux-en-Y gastric bypass (LRYGB) with 100-cm alimentary and 50-cm biliopancreatic limb in a 40-year-old morbidly obese patient with a history of shortness of breath on exertion, carpal tunnel syndrome, and joint disease, who

was admitted for surgery on May 31, 2007. The procedure was performed without apparent complications. His postoperative course was unremarkable; on postoperative day 1, the patient successfully passed a Gastrografin swallow study showing no leak or extravasations and was subsequently advanced to a phase 1 liquid diet. The patient tolerated a liquid diet well, and by postoperative day 3, he was afebrile. Lab work was within normal values, and his vital signs were stable. His Jackson–Pratt drains were discontinued prior to discharge. On August 2008, an abdominal CT with contrast was ordered for mild abdominal pain and nausea, showing small amount of fluid at the level of the liver, spleen, and right paracolic gutter as well as mesenteric adenitis (Fig. 1).

On September 2008, the patient is admitted to the Cleveland Clinic Florida due to chronic abdominal pain of unknown etiology. At physical exam, the abdomen was soft with generalized mild, tender, not distended with normal bowel sounds. Subocclusion symptoms were not present. The patient is schedule for an elective diagnostic laparoscopy and possible reduction of internal hernia and closure of a mesenteric defect. Upon inspection of the abdominal cavity, it comes to our attention that there is a large amount of chylous fluid accumulating in the right upper quadrant, cul-de-sac, and left upper quadrant (Figs. 2 and 3); a specimen was sent for microscopic analysis, which reported high triglyceride content (fat) and negative for any bacteria or lymphocytes (Fig. 4).

An internal herniation of the common channel was also found (Fig. 5). The common channel was run from the ileocecal valve to the jejunojejunal anastomosis reducing the internal herniation. In addition, an adhesive band that is crossing the alimentary limb is divided with a harmonic scalpel. The jejunojejunal mesenteric defect is then suture-closed with a running 2-0 silk suture.

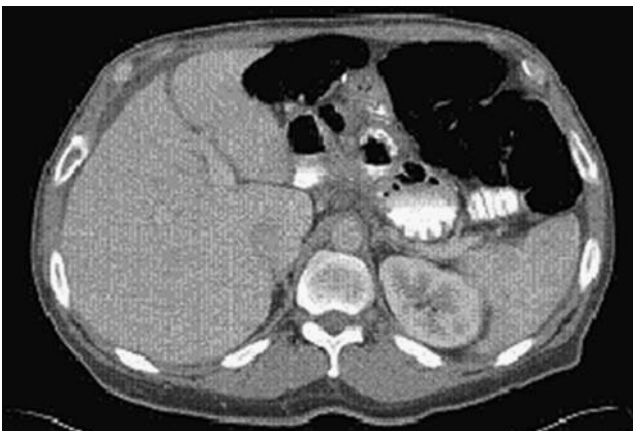


Fig. 1 Abdominal CT with contrast showing small amount of fluid at liver, spleen, right paracolic gutter, and mesenteric adenitis



Fig. 2 Image showing chylous fluid accumulating in the right upper quadrant, cul-de-sac, and left upper quadrant

The patient made an uneventful recovery after surgery. To our knowledge, this is the first reported case of chylous ascites following a LRYGB.

Discussion

The evaluation of a patient with ascites requires that the cause of the ascites be established. In most cases, ascites appears as part of a well-recognized illness, i.e., cirrhosis, congestive heart failure, nephrosis, or disseminated carcinomatosis. In these situations, the physician should determine that the development of ascites is indeed a consequence of the basic underlying disease and not due to the presence of a separate or related disease process. This distinction is necessary even when the cause of ascites seems obvious. For example, when the patient with compensated cirrhosis and minimal ascites develops progressive ascites that is increasingly difficult to control with



Fig. 3 Image showing chylous fluid accumulating in the right upper quadrant, cul-de-sac, and left upper quadrant



Fig. 4 Image of a specimen reported to have high triglyceride content

sodium restriction or diuretics, the temptation is to attribute the worsening of the clinical picture to progressive liver disease. However, an occult hepatocellular carcinoma, portal vein thrombosis, spontaneous bacterial peritonitis, alcoholic hepatitis, viral infection, or even tuberculosis may be responsible for the decompensation. The disappointingly low success in diagnosing tuberculous peritonitis or hepatocellular carcinoma in a patient with cirrhosis and ascites reflects the too-low index of suspicion for the development of such superimposed conditions. Similarly, the patient with congestive heart failure may develop ascites from a disseminated carcinoma with peritoneal seeding. It is important to note, however, that while there are many different causes of ascites, in the USA, >80% of cases are due to cirrhosis [4].

The typical presentation of chylous ascites is abdominal distention and pain along with vague constitutional symptoms. Physical findings—besides ascites—include concomitant pleural effusion and peripheral edema. The combination of fever, night sweats, and lymphadenopathy should arouse suspicion of a lymphoma. About 15% of cases occur in young children (usually <1 year old) with congenital lymphatic anomalies. Patients with chylous ascites develop

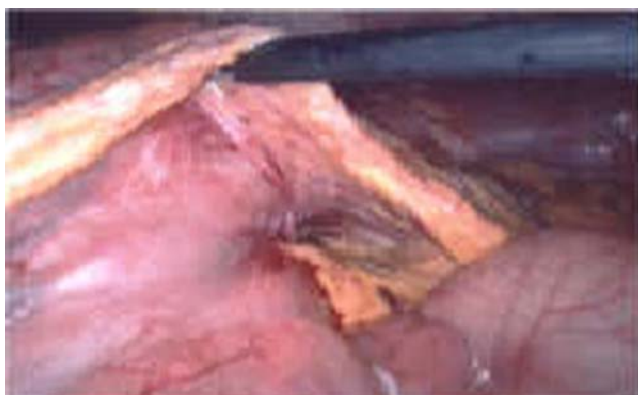


Fig. 5 Image showing internal herniation of the common channel

abdominal distention over a period of weeks to months. Postoperative chylous ascites can present acutely during the first postoperative week. Delayed presentations following surgery can occur if the mechanism of ascites formation is adhesion-induced lymphatic obstruction rather than lymphatic vessel disruption. Dyspnea may result if abdominal distention is severe enough [5].

Chylous ascites caused by intestinal lymphatic obstruction is associated with fat malabsorption and protein loss. Intestinal loss of albumin and γ -globulin may lead to edema and increase the risk of infection. Rapidly accumulating chylous ascites may cause respiratory complications [6].

Paracentesis is the most important diagnostic test. Chyle typically has a turbid appearance; however, it may be clear in fasting patients (such as those in the immediate postoperative period). Fluid triglyceride concentrations above 110 mg/dl are diagnostic. CT scanning may be useful in identifying pathologic intra-abdominal lymph nodes and masses and in identifying extent and localization of fluid. Lymphangiography and lymphoscintigraphy may help to localize lymph leaks and obstruction; this information is particularly useful for surgical planning. Conventional radiologic investigations, particularly CT scan of the abdomen, may be helpful. Lymph node biopsy, where applicable, and laparotomy have the highest diagnostic value [5].

Management of patients with chylous ascites should focus on evaluating and treating the underlying causes, especially for patients with infectious, inflammatory, or hemodynamic etiologies for this condition. Most patients respond to administration of a high-protein and low-fat diet supplemented with medium-chain triglycerides. This regimen is designed to minimize chyle production and flow. Medium-chain triglycerides are absorbed by the intestinal epithelium and are transported to the liver through the portal vein; they do not contribute to chylomicron formation. Patients who do not respond to this approach should be fasted and placed on total parenteral nutrition. Octreotide can further decrease lymph flow. Paracentesis is indicated for respiratory difficulties related to abdominal distention. Overall, more than 60% of patients will respond to conservative therapy. However, approximately 30% of patients will require surgical therapy for chylous ascites. In general, postoperative and trauma-related cases that fail to respond to initial nonoperative therapy are best managed by surgical repair. Lymphatic leaks are localized and repaired with fine nonabsorbable sutures. If extravasation of chyle is localized to the periphery of the small-bowel mesentery, then a limited small-bowel resection can be performed instead. For patients who are poor surgical candidates and who do not respond to prolonged conservative therapy, peritoneovenous shunting may be an option.

However, these shunts are associated with high rates of complications, including sepsis and disseminated intravascular coagulation. Because of the viscosity of chyle, these shunts are associated with a high occlusion rate.

An analysis of the physiologic and metabolic factors involved in the production of ascites, coupled with a complete evaluation of the nature of the ascitic fluid, invariably discloses the etiology of the ascites and permits appropriate therapy to be instituted [4].

Conclusion

Chyloperitoneum should be considered as a possible cause of ascites in patients with chronic small bowel obstruction following a LRYGB.

References

1. Krizek TJ, Davis JH. Acute chylous peritonitis. *Arch Surg.* 1965;91:253–62.
2. Madding GF, McLaughlin RF, McLaughlin RF Jr. Acute chylous peritonitis. *Ann Surg.* 1958;147(3):419–22.
3. Doherty GM. Peritoneal cavity, Chapter 22. In: Doherty GM, Way LW, editors. *Current surgical diagnosis and treatment.* 12th ed. New York: McGraw-Hill; 2005.
4. Whang EE, Ashley SW, Zinner MJ. Small intestine, Chapter 27. In: Brunnicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE, Schwartz SI, editors. *Schwartz's principles of surgery.* 8th ed. New York: McGraw-Hill; 2004.
5. Glickman RM, Rajapaksa R. Abdominal swelling and ascites, Chapter 44. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J, editors. *Harrison's principles of internal medicine.* 17th ed. New York: McGraw-Hill; 2008.
6. Sondheimer JM, Sundaram S. Gastrointestinal tract, Chapter 20. In: Hay Jr WW, Levin MJ, Sondheimer JM, editors. *Current pediatric diagnosis and treatment.* New York: Mc-Graw-Hill; 2005.